

=> d his

(FILE 'HOME' ENTERED AT 10:51:48 ON 24 APR 2002)

FILE 'HCAPLUS' ENTERED AT 10:52:32 ON 24 APR 2002

L1 65 S LAHIRI J?/AU
 L2 2088 S FANG Y?/AU
 L3 122 S JONAS S?/AU
 L4 19 S KALAL P?/AU
 L5 10759 S WANG W?/AU
 L6 13029 S L1-5
 L7 526 S L6 AND ?MEMBRANE?
 L8 27 S L7 AND ASSAY?
 L9 3 S L8 AND (CHIP OR ?ARRAY? OR SURFACE OR ?SILAN? OR GLASS)
 L10 24 S L8 NOT L9
 L11 1 S L10 AND LIGAND(2A)BIND?
 L12 4 S L9 OR L11
 SELECT RN L12 1-4

- invento
Search

'FILE 'REGISTRY' ENTERED AT 10:59:10 ON 24 APR 2002

L13 4 S E1-4

FILE 'HCAPLUS' ENTERED AT 10:59:32 ON 24 APR 2002

L14 2 S L12 AND L13
 L15 4 S L12 OR L14 4 citations for inventors
 L16 509 S (?PROTEIN? OR ?PEPTID?) (2A) CHIP
 L17 1 S L16 AND L6
 L18 24061 S (?PROTEIN? OR ?PEPTID?) (5A) (?IMMOB? OR ATTACH? OR SPAN? OR FI
 L19 18 S L6 AND L18
 L20 9 S L19 AND (?ARRAY? OR SURFACE OR ?SILAN? OR GLASS OR ?MEMBRAN?)
 L21 9 S L20 NOT L15
 L22 10 S L17 OR L21 10 cites for inventors

FILE 'REGISTRY' ENTERED AT 11:15:25 ON 24 APR 2002

L23 141484 S "SILANE"
 L24 25828 S "AMINOPROPYL"
 L25 569 S L23 AND L24
 L26 220 S L25 AND "GAMMA"
 L27 57 S L26 AND NC=1 NOT PMS/CI
 L28 43 S L27 NOT RSD/FA
 L29 3 S L28 NOT O/ELS
 L30 1 S L29 AND C3 H11 N SI/MF claim 51 cpd
 L31 0 S 6382-82-7 /CRN

FILE 'HCAPLUS' ENTERED AT 11:21:53 ON 24 APR 2002

L32 3 S L30/PREP 3 cites for prep. of 8-aminoethylsilane
 FILE 'CASREACT' ENTERED AT 11:25:07 ON 24 APR 2002
 L33 STR 6382-82-7
 L34 0 S L33
 L35 0 S L33 FUL no cites for prep of ↑

FILE 'HCAPLUS' ENTERED AT 12:24:50 ON 24 APR 2002

L36 156729 S L16 OR L18 OR (?PROTEIN? OR ?PEPTID?) (5A) (?ASSOCIAT? OR ?COU
 L37 668665 S ?MEMBRAN? OR ?BILAYER? OR ?AMPHIPHILIC?(3A)?SURFAC? OR ?PROT
 L38 289077 S ?PRINT? OR QUILL? OR QUILL-PIN OR SPOT? OR MICROSPOT?
 L39 1041533 S GLASS OR SILICA OR QUARTZ
 L40 73 S L30
 L41 43272 S G-PROTEIN
 L42 349 S L36 AND L37 AND L38

L43 1 S L42 AND L40
 E HIW
 L44 1 S L39 AND L43
 L45 1 S L44 AND L41 1 cite
 L46 14 S L42 AND L39
 L47 2 S L46 AND L41
 L48 1 S L47 NOT (L45 OR L12) 1 cite
 L49 12 S L46 NOT L47-48
 L50 5 S L49 AND PATENT/DT
 L51 4 S L50 AND PRD<20000810
 L52 7 S L49 NOT L50
 L53 5 S L52 AND PD<20000810
 L54 9 S L51 OR L53 9 cites
 L55 11 S L39 AND L38 AND L41
 L56 9 S L55 NOT (L46 OR L12 OR L48)
 L57 3 S L56 AND L36-37 3 cites
 L58 349 S L36 AND L37 AND L38
 L59 5 S L58 AND CHIP
 L60 16 S L58 AND MICROARRAY
 L61 19 S L59-60
 L62 4 S L61 AND L41
 L63 2 S L62 NOT (L46 OR L12 OR L48) 2 cites
 L64 4 S L61 AND L39
 L65 3 S L64 NOT L62-63
 L66 2 S L65 AND PRD<20000810 2 patents
 L67 2 S L40 AND L38
 L68 4 S L40 AND (L38 OR L41 OR L36-38)
 L69 3 S L68 NOT (L46 OR L12 OR L48) 3 cites
 L70 9141 S L36(P)ASSAY?
 L71 841989 S PROBE OR TARGET OR CONTACT
 L72 1244 S L70 AND L71
 L73 295 S L70 AND (CONTACT? OR SPOT? OR MICROSPOT?)
 L74 87 S L73 AND DETECT?
 L75 12 S L74 AND TARGET
 L76 9 S L74 AND PROBE
 L77 4 S L75 AND L76
 L78 16 S L74 AND SOLUTION
 L79 2 S L77 AND L78 2 sites
 L80 30123 S L37 (P)ASSAY?
 L81 2210 S L70 AND L80
 L82 2170 S L70 (P)L80
 L83 63 S L82(P) (CONTACT? OR SPOT? OR MICROSPOT? OR PIN)
 L84 6 S L83(P) (TARGET OR PROBE)
 L85 0 S L83 AND (L39 OR SIO2)
 L86 13 S L82 AND (L39 OR SIO2)
 L87 0 S L86 AND (CONTACT? OR SPOT? OR MICROSPOT? OR PIN OR TIP)
 L88 80 S L81 AND (CONTACT? OR SPOT? OR MICROSPOT? OR PIN OR TIP)
 L89 2 S L88 AND (SIO2 OR L39)
 L90 19 S L88 AND SURFACE
 L91 477 S CHIP(P)ASSAY?
 L92 126 S L91(P)SURFACE
 L93 11 S L92(P)?MEMBRANE? 11 cites
 L94 18 S (PROTEIN OR ?PEPTID?) (5A)CHIP(10A)PREPAR?
 L95 0 S L94 AND PIN
 L96 4 S L94 AND ?MEMBRAN?
 L97 0 S L94 AND BLOT?
 L98 1 S L94 AND (?PATTERN? OR ?SPOT?)
 L99 4 S L96 OR L98
 L100 3 S L99 NOT (L46 OR L12 OR L48) 3 cites

Inventor Search

TRAN 09/854,786

=> d que l15

L1	65 SEA FILE=HCAPLUS ABB=ON	PLU=ON	LAHIRI J?/AU
L2	2088 SEA FILE=HCAPLUS ABB=ON	PLU=ON	FANG Y?/AU
L3	122 SEA FILE=HCAPLUS ABB=ON	PLU=ON	JONAS S?/AU
L4	19 SEA FILE=HCAPLUS ABB=ON	PLU=ON	KALAL P?/AU
L5	10759 SEA FILE=HCAPLUS ABB=ON	PLU=ON	WANG W?/AU
L6	13029 SEA FILE=HCAPLUS ABB=ON	PLU=ON	(L1 OR L2 OR L3 OR L4 OR L5)
L7	526 SEA FILE=HCAPLUS ABB=ON	PLU=ON	L6 AND ?MEMBRANE?
L8	27 SEA FILE=HCAPLUS ABB=ON	PLU=ON	L7 AND ASSAY?
L9	3 SEA FILE=HCAPLUS ABB=ON	PLU=ON	L8 AND (CHIP OR ?ARRAY? OR SURFACE OR ?SILAN? OR GLASS)
L10	24 SEA FILE=HCAPLUS ABB=ON	PLU=ON	L8 NOT L9
L11	1 SEA FILE=HCAPLUS ABB=ON	PLU=ON	L10 AND LIGAND(2A)BIND?
L12	4 SEA FILE=HCAPLUS ABB=ON	PLU=ON	L9 OR L11
L13	4 SEA FILE=REGISTRY ABB=ON	PLU=ON	(127361-24-4/B1 OR 217962-21-5/B1 OR 250154-17-7/B1 OR 80451-05-4/B1)
L14	2 SEA FILE=HCAPLUS ABB=ON	PLU=ON	L12 AND L13
L15	4 SEA FILE=HCAPLUS ABB=ON	PLU=ON	L12 OR L14

4 cites

=> d ibib abs hitstr 1

L15 ANSWER 1 OF 4 HCPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:145010 HCPLUS
TITLE: **Membrane Protein Microarrays**
AUTHOR(S): Fang, Ye; Frutos, Anthony G.; Lahiri,
Joydeep
CORPORATE SOURCE: Biochemical Technologies, Science and Technology
Division, Corning Incorporated, Corning, NY, 14831,
USA
SOURCE: Journal of the American Chemical Society (2002),
124(11), 2394-2395
CODEN: JACSAT; ISSN: 0002-7863
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal
LANGUAGE: English
AB This paper describes the fabrication of **microarrays** consisting
of G protein-coupled receptors (GPCRs) on **surfaces** coated with
.gamma.-**aminopropylsilane** (GAPS). Microspots of model
membranes on GAPS-coated **surfaces** were obsd. to have
several desired properties-high mech. stability, long range lateral
fluidity, and a thickness corresponding to a lipid bilayer in the bulk of
the microspot. GPCR **arrays** were obtained by printing
membrane prepns. contg. GPCRs using a quill-pin printer. To
demonstrate specific binding of ligands, **arrays** presenting
neurotensin (NTR1), adrenergic (.beta.1), and dopamine (D1) receptors were
treated with fluorescently labeled neurotensin (BT-NT). Fluorescence
images revealed binding only to microspots corresponding to the
neurotensin receptor; this specificity was further demonstrated by the
inhibition of binding in the presence of excess unlabeled neurotensin.
The ability of GPCR **arrays** to enable selectivity studies between
the different subtypes of a receptor was examd. by printing **arrays**
consisting of three subtypes of the adrenergic receptor: .beta.1,
.beta.2, and .alpha.2A. When treated with fluorescently labeled CGP
12177, a cognate antagonist analog specific to .beta.-adrenergic
receptors, binding was only obsd. to microspots of the .beta.1 and .beta.2
receptors. Furthermore, binding of labeled CGP 12177 was inhibited when
the **arrays** were incubated with solns. also contg. ICI 118551,
and in a manner consistent with the higher affinity of ICI 118551 for the
.beta.2 receptor relative to that for the .beta.1 receptor. The ability
to est. binding affinities of compds. using GPCR **arrays** was
examd. using a competitive binding **assay** with BT-NT and
unlabeled nuerotensin on NTR1 **arrays**. The estd. IC50 value (2
nM) for neurotensin is in agreement with the literature; this agreement
suggests that the receptor-G protein complex is preserved in the
microspot. This first ever demonstration of direct pin-printing of
membrane proteins and ligand-binding **assays** thereof
fills a significant void in protein microchip technol.-the lack of
practical **microarray**-based methods for **membrane**
proteins.

REFERENCE COUNT: 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

TRAN 09/854,786

=> d ind 1

L15 ANSWER 1 OF 4 HCAPLUS COPYRIGHT 2002 ACS
CC 9 (Biochemical Methods)

=> d ibib abs hitstr 2

L15 ANSWER 2 OF 4 HCPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 2001:230792 HCPLUS
 DOCUMENT NUMBER: 134:363049
 TITLE: Kinetics of **membrane** lysis by custom lytic peptides and peptide orientations in **membrane**
 AUTHOR(S): Chen, H. M.; Clayton, A. H. A.; Wang, W.; Sawyer, W. H.
 CORPORATE SOURCE: Department of Biochemistry, Hong Kong University of Science and Technology, Kowloon, Hong Kong
 SOURCE: European Journal of Biochemistry (2001), 268(6), 1659-1669
 CODEN: EJBCAI; ISSN: 0014-2956
 PUBLISHER: Blackwell Science Ltd.
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB To aid the development of custom peptide antibiotics, a kinetic study of **membrane** lysis by cecropin B (CB) and its analogs, cecropin B1 (CB1) and cecropin B3 (CB3) was carried out to det. the mechanism by which these peptides disrupt the bilayer structure of liposomes of defined compn. Disruption of the phospholipid bilayer was detd. by a fluorescence assay involving the use of dithionite to quench the fluorescence of lipids labeled with N-7-nitro-2,1,3-benzoxadiazol-4-yl. Lytic peptides caused the disruption of liposomes to occur in two kinetic steps. For liposomes composed of mixts. of phosphatidylcholine and phosphatidic acid, the time consts. for each kinetic step were shorter for CB and CB1 than for CB3. Oriented CD expts. showed that the peptides could exist in at least two different **membrane**-assocd. states that differed primarily in the orientation of the helical segments with respect to the bilayer **surface**. The results are discussed in terms of kinetic mechanisms of **membrane** lysis. The mode of actions of these peptides used for the interpretation of their kinetic mechanisms were supported by **surface** plasmon resonance expts. including or excluding the pore-forming activities.
 IT 80451-05-4, Cecropin B 127361-24-4, Cecropin B1
 217962-21-5, Cecropin B3
 RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
 (kinetics of **membrane** lysis by custom lytic peptides and peptide orientations in **membrane** using fluorescence-quenching and oriented CD spectroscopy)
 RN 80451-05-4 HCPLUS
 CN Cecropin B (Platysamia cecropia antibacterial peptide) (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

RN 127361-24-4 HCPLUS
 CN L-Lysinamide, L-lysyl-L-tryptophyl-L-lysyl-L-valyl-L-phenylalanyl-L-lysyl-L-lysyl-L-isoleucyl-L-.alpha.-glutamyl-L-lysyl-L-methionylglycyl-L-arginyl-L-asparaginyl-L-isoleucyl-L-arginyl-L-asparaginylglycyl-L-isoleucyl-L-valyl-L-lysyl-L-alanylglycyl-L-prolyl-L-lysyl-L-tryptophyl-L-lysyl-L-valyl-L-phenylalanyl-L-lysyl-L-lysyl-L-isoleucyl-L-.alpha.-glutamyl- (9CI) (CA INDEX NAME)

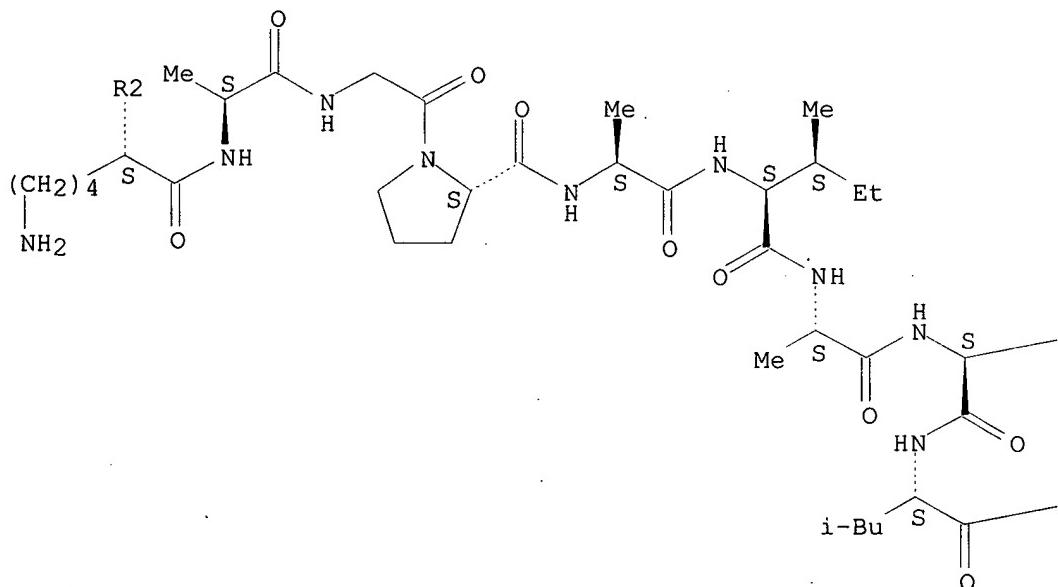
*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

RN 217962-21-5 HCPLUS
 CN L-Leucinamide, L-alanyl-L-isoleucyl-L-alanyl-L-valyl-L-leucylglycyl-L-.alpha.-glutamyl-L-alanyl-L-lysyl-L-alanyl-L-leucyl-L-methionylglycyl-L-

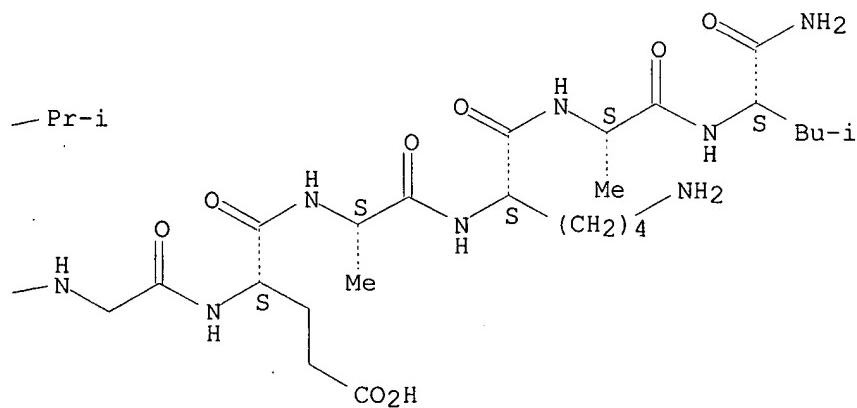
arginyl-L-asparaginyl-L-isoleucyl-L-arginyl-L-asparaginylglycyl-L-isoleucyl-L-valyl-L-lysyl-L-alanylglycyl-L-prolyl-L-alanyl-L-isoleucyl-L-alanyl-L-valyl-L-leucylglycyl-L-.alpha.-glutamyl-L-alanyl-L-lysyl-L-alanyl-(9CI) (CA INDEX NAME)

Absolute stereochemistry.

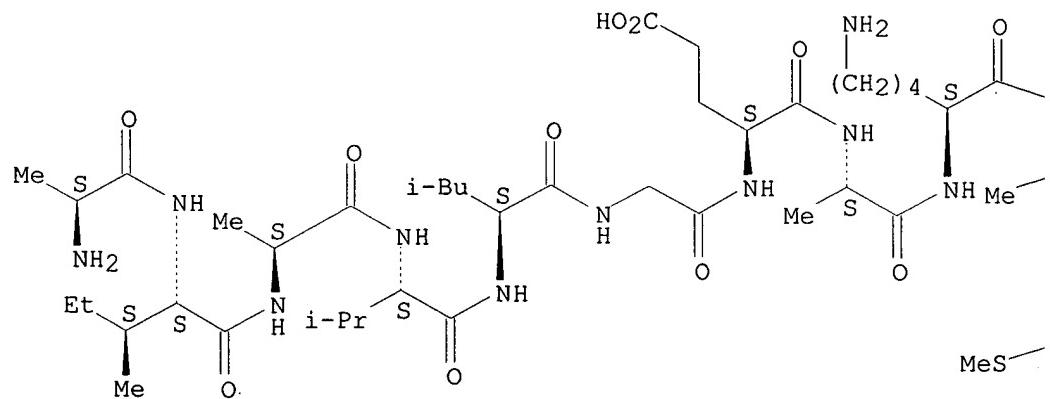
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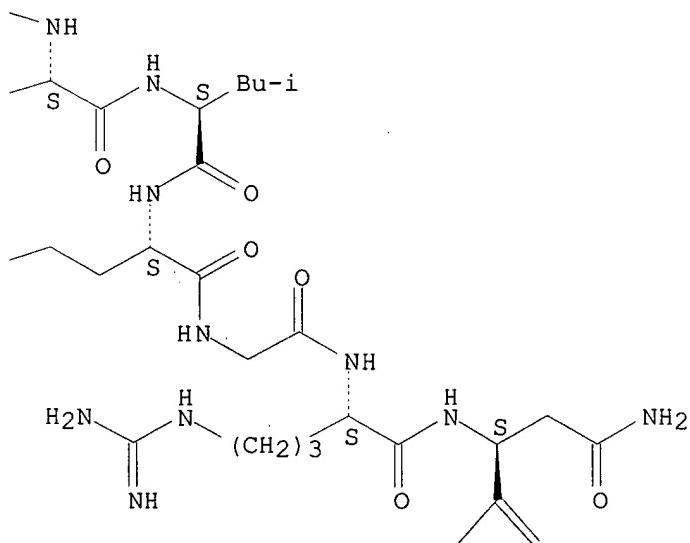
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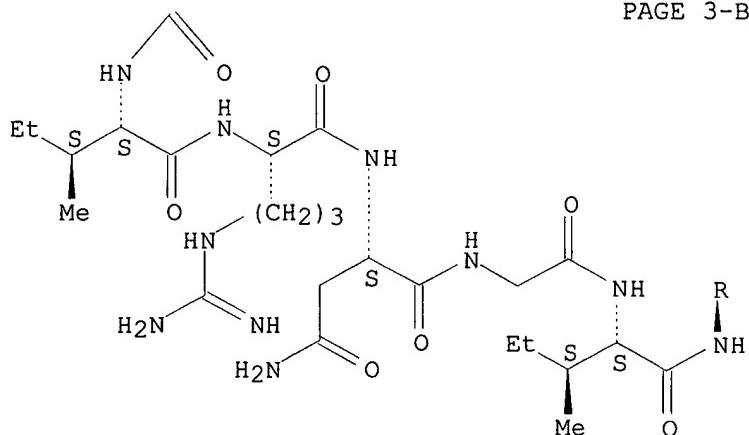
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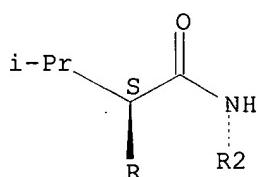
PAGE 2-B



PAGE 3-B



PAGE 4-A



REFERENCE COUNT:

31

THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d ind 2

L15 ANSWER 2 OF 4 HCPLUS COPYRIGHT 2002 ACS
CC 6-6 (General Biochemistry)
ST kinetics **membrane** lysis peptide
IT **Membrane**, biological
(bilayer; kinetics of **membrane** lysis by custom lytic peptides
and peptide orientations in **membrane** using
fluorescence-quenching and oriented CD spectroscopy)
IT Physical process kinetics
(**membrane** lysis; kinetics of **membrane** lysis by
custom lytic peptides and peptide orientations in **membrane**
using fluorescence-quenching and oriented CD spectroscopy)
IT Phosphatidic acids
Phosphatidylcholines, biological studies
Phospholipids, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL
(Biological study); PROC (Process)
(**membrane**; kinetics of **membrane** lysis by custom
lytic peptides and peptide orientations in **membrane** using
fluorescence-quenching and oriented CD spectroscopy)
IT Conformation
(protein; kinetics of **membrane** lysis by custom lytic peptides
and peptide orientations in **membrane** using
fluorescence-quenching and oriented CD spectroscopy)
IT 80451-05-4, Cecropin B 127361-24-4, Cecropin B1
217962-21-5, Cecropin B3
RL: BAC (Biological activity or effector, except adverse); BSU (Biological
study, unclassified); PRP (Properties); BIOL (Biological study)
(kinetics of **membrane** lysis by custom lytic peptides and
peptide orientations in **membrane** using fluorescence-quenching
and oriented CD spectroscopy)

=> d ibib abs hitstr 3

L15 ANSWER 3 OF 4 HCPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:5303 HCPLUS
DOCUMENT NUMBER: 132:133833
TITLE: Affinity purification and partial characterization of the zonulin/zonula occludens toxin (Zot) receptor from human brain
AUTHOR(S): Lu, R.; Wang, W.; Uzzau, S.; Vigorito, R.; Zielke, H. R.; Fasano, A.
CORPORATE SOURCE: Division of Pediatric Gastroenterology and Nutrition and Center for Vaccine Development, University of Maryland School of Medicine, Baltimore, MD, 21201, USA
SOURCE: Journal of Neurochemistry (2000), 74(1), 320-326
CODEN: JONRA9; ISSN: 0022-3042
PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The intercellular tight junctions (TJs) of endothelial cells represent the limiting structure for the permeability of the blood-brain barrier (BBB). Although the BBB has been recognized as being the interface between the bloodstream and the brain, little is known about its regulation. Zonulin and its prokaryotic analog, zonula occludens toxin (Zot) elaborated by *Vibrio cholerae*, both modulate intercellular TJs by binding to a sp. surface receptor with subsequent activation of an intracellular signaling pathway involving phospholipase C and protein kinase C activation and actin polymn. Affinity column purifn. revealed that human brain plasma membrane preps. contain two Zot binding proteins of .apprx.55 and .apprx.45 kDa. Structural and kinetic studies, including satn. and competitive assays, identified the 55-kDa protein as tubulin, whereas the 45-kDa protein represents the zonulin/Zot receptor. Biochem. characterization provided evidence that this receptor is a glycoprotein contg. multiple sialic acid residues. Comparison of the N-terminal sequence of the zonulin/Zot receptor with other protein sequences by BLAST anal. revealed a striking similarity with MRP-8, a 14-kDa member of the S-100 family of calcium binding proteins. The discovery and characterization of this receptor from human brain may significantly contribute to our knowledge on the pathophysiol. regulation of the BBB.
REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d ind 3

L15 ANSWER 3 OF 4 HCAPLUS COPYRIGHT 2002 ACS
CC 6-3 (General Biochemistry)
Section cross-reference(s): 4, 13
ST zonulin zonula occludens toxin receptor protein brain human
IT Proteins, specific or class
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(MRP-8 (migration-inhibiting factor-related, 8000-mol.-wt.); affinity
purifn. and partial characterization of zonulin/zonula occludens toxin
(Zot) receptor from human brain)
IT Proteins, specific or class
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological
study, unclassified); PRP (Properties); BIOL (Biological study); OCCU
(Occurrence); PROC (Process)
(Zot binding protein; affinity purifn. and partial characterization of
zonulin/zonula occludens toxin (Zot) receptor from human brain)
IT Blood-brain barrier
Brain
Heart
Intestine
Protein sequences
(affinity purifn. and partial characterization of zonulin/zonula
occludens toxin (Zot) receptor from human brain)
IT Cell junction
(tight junction; affinity purifn. and partial characterization of
zonulin/zonula occludens toxin (Zot) receptor from human brain)
IT Receptors
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological
study, unclassified); PRP (Properties); BIOL (Biological study); OCCU
(Occurrence); PROC (Process)
(zonulin/zonula occludens toxin; affinity purifn. and partial
characterization of zonulin/zonula occludens toxin (Zot) receptor from
human brain)
IT Toxins
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL
(Biological study); PROC (Process)
(zonulin/zonula occludens toxin; affinity purifn. and partial
characterization of zonulin/zonula occludens toxin (Zot) receptor from
human brain)

=> d ibib abs hitstr 4

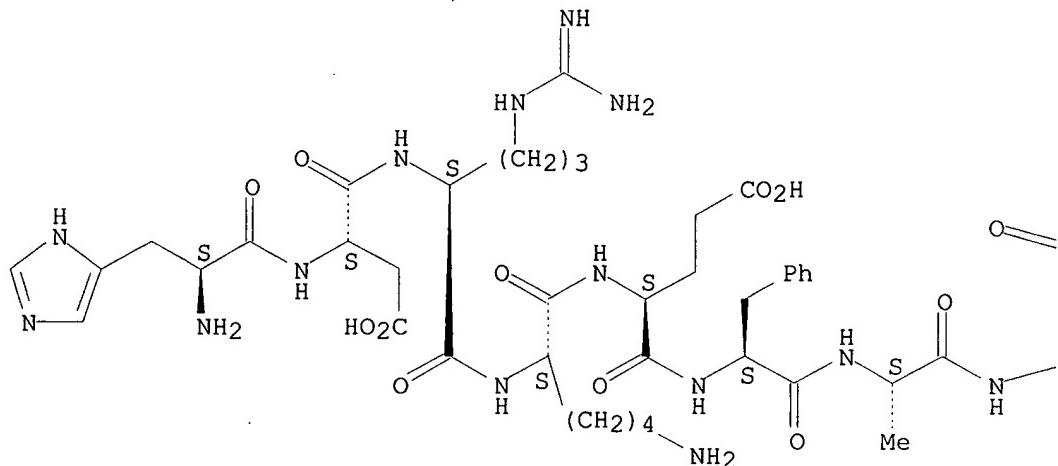
L15 ANSWER 4 OF 4 HCPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1999:651397 HCPLUS
 DOCUMENT NUMBER: 131:333706
 TITLE: Identification of a talin-binding site in the integrin .beta.3 subunit distinct from the NPLY regulatory motif of post-ligand binding functions. The talin N-terminal head domain interacts with the membrane-proximal region of the .beta.3 cytoplasmic tail
 AUTHOR(S): Patil, Sonali; Jedsadayanmata, Arom; Wencel-Drake, June D.; Wang, Wei; Knezevic, Irina; Lam, Stephen C.-T.
 CORPORATE SOURCE: Department of Pharmacology, The University of Illinois, Chicago, IL, 60612, USA
 SOURCE: J. Biol. Chem. (1999), 274(40), 28575-28583
 CODEN: JBCHA3; ISSN: 0021-9258
 PUBLISHER: American Society for Biochemistry and Molecular Biology
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB Following platelet aggregation, integrin .alpha.IIb.beta.3 becomes assocd. with the platelet cytoskeleton. The conserved NPLY sequence represents a potential .beta.-turn motif in the .beta.3 cytoplasmic tail and has been suggested to mediate the interaction of .beta.3 integrins with talin. In the present study, the authors performed a double mutation (N744Q/P745A) in the integrin .beta.3 subunit to test the functional significance of this .beta.-turn motif. Chinese hamster ovary cells were co-transfected with cDNA constructs encoding mutant .beta.3 and wild type .alpha.IIb. Cells expressing either wild type (A5) or mutant (D4) .alpha.IIb.beta.3 adhered to fibrinogen; however, as opposed to control A5 cells, adherent D4 cells failed to spread, form focal adhesions, or initiate protein tyrosine phosphorylation. To investigate the role of the NPLY motif in talin binding, the authors exmd. the ability of the mutant .alpha.IIb.beta.3 to interact with talin in a solid phase binding assay. Both wild type and mutant .alpha.IIb.beta.3, purified by RGD affinity chromatog., bound to a similar extent to immobilized talin. Addnl., purified talin failed to interact with peptides contg. the AKWDTANNPLYK sequence indicating that the talin binding domain in the integrin .beta.3 subunit does not reside in the NPLY motif. In contrast, specific binding of talin to peptides contg. the membrane -proximal HDRKEFAKFEEERARAK sequence of the .beta.3 cytoplasmic tail was obsd., and this interactions was blocked by a recombinant protein fragment corresponding to the 47-kDa N-terminal head domain of talin (rTalin-N). In addn., RGD affinity purified platelet .alpha.IIb.beta.3 bound dose-dependently to immobilized rTalin-N, indicating that an integrin-binding site is present in the talin N-terminal head domain. Collectively, these studies demonstrate that the NPLY .beta.-turn motif regulates post-ligand binding functions of .alpha.IIb.beta.3 in a manner independent of talin interaction. Moreover, talin was shown to bind through its N-terminal head domain to the membrane-proximal sequence of the .beta.3 cytoplasmic tail.
 IT 250154-17-7
 RL: BOC (Biological occurrence); BPR (Biological process); PRP (Properties); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
 (as talin N-terminal head domain-binding site in integrin .beta.3 subunit membrane-proximal cytoplasmic tail region)
 RN 250154-17-7 HCPLUS

TRAN 09/854, 786

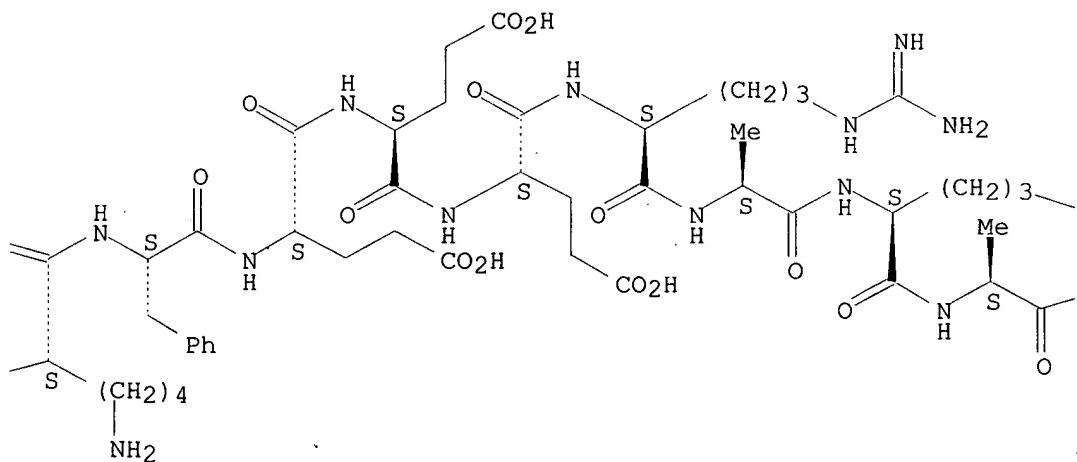
CN L-Lysine, L-histidyl-L-.alpha.-aspartyl-L-arginyl-L-lysyl-L-.alpha.-glutamyl-L-phenylalanyl-L-alanyl-L-lysyl-L-phenylalanyl-L-.alpha.-glutamyl-L-.alpha.-glutamyl-L-.alpha.-glutamyl-L-arginyl-L-alanyl-L-arginyl-L-alanyl- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

PAGE 1-A

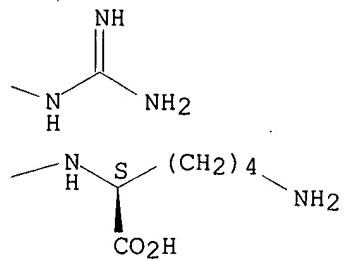


PAGE 1-B



TRAN 09/854, 786

PAGE 1-C



REFERENCE COUNT:

60

THERE ARE 60 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d ind 4

L15 ANSWER 4 OF 4 HCPLUS COPYRIGHT 2002 ACS
 CC 6-3 (General Biochemistry)
 ST talin N terminal head domain binding site integrin beta3
 IT Fibrinogens
 RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
 (adhesion to; talin N-terminal head domain-binding site in integrin
 .beta.3 subunit **membrane**-proximal cytoplasmic tail region
 distinct from NPLY .beta.-turn regulatory motif of post-**ligand**
 binding functions in relation to)
 IT Platelet (blood)
 (aggregation; talin N-terminal head domain-binding site in integrin
 .beta.3 subunit **membrane**-proximal cytoplasmic tail region
 distinct from NPLY .beta.-turn regulatory motif of post-**ligand**
 binding functions in relation to)
 IT Cell junction
 (focal contact; talin N-terminal head domain-binding site in integrin
 .beta.3 subunit **membrane**-proximal cytoplasmic tail region
 distinct from NPLY .beta.-turn regulatory motif of post-**ligand**
 binding functions in relation to)
 IT Cell aggregation
 (platelet; talin N-terminal head domain-binding site in integrin
 .beta.3 subunit **membrane**-proximal cytoplasmic tail region
 distinct from NPLY .beta.-turn regulatory motif of post-**ligand**
 binding functions in relation to)
 IT Phosphorylation, biological
 (protein; talin N-terminal head domain-binding site in integrin .beta.3
 subunit **membrane**-proximal cytoplasmic tail region distinct
 from NPLY .beta.-turn regulatory motif of post-**ligand**
 binding functions in relation to)
 IT Protein motifs
 (talin N-terminal head domain-binding site in integrin .beta.3 subunit
 membrane-proximal cytoplasmic tail region distinct from NPLY
 .beta.-turn regulatory motif of post-**ligand** **binding**
 functions)
 IT Talin
 RL: BPR (Biological process); PRP (Properties); BIOL (Biological study);
 PROC (Process)
 (talin N-terminal head domain-binding site in integrin .beta.3 subunit
 membrane-proximal cytoplasmic tail region distinct from NPLY
 .beta.-turn regulatory motif of post-**ligand** **binding**
 functions)
 IT Integrins
 RL: BPR (Biological process); PRP (Properties); BIOL (Biological study);
 PROC (Process)
 (.alpha.IIb.beta.3; talin N-terminal head domain-binding site in
 integrin .beta.3 subunit **membrane**-proximal cytoplasmic tail
 region distinct from NPLY .beta.-turn regulatory motif of post-
 ligand **binding** functions)
 IT Integrins
 RL: BPR (Biological process); PRP (Properties); BIOL (Biological study);
 PROC (Process)
 (.beta.3; talin N-terminal head domain-binding site in integrin .beta.3
 subunit **membrane**-proximal cytoplasmic tail region distinct
 from NPLY .beta.-turn regulatory motif of post-**ligand**
 binding functions)
 IT 250154-17-7
 RL: BOC (Biological occurrence); BPR (Biological process); PRP

TRAN 09/854,786

(Properties); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(as talin N-terminal head domain-binding site in integrin .beta.3
subunit **membrane**-proximal cytoplasmic tail region)

Inventor ~~see~~
search

TRAN 09/854,786

=> d que 122

L1 65 SEA FILE=HCAPLUS ABB=ON PLU=ON LAHIRI J?/AU
L2 2088 SEA FILE=HCAPLUS ABB=ON PLU=ON FANG Y?/AU
L3 122 SEA FILE=HCAPLUS ABB=ON PLU=ON JONAS S?/AU
L4 19 SEA FILE=HCAPLUS ABB=ON PLU=ON KALAL P?/AU
L5 10759 SEA FILE=HCAPLUS ABB=ON PLU=ON WANG W?/AU
L6 13029 SEA FILE=HCAPLUS ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5)
L7 526 SEA FILE=HCAPLUS ABB=ON PLU=ON L6 AND ?MEMBRANE?
L8 27 SEA FILE=HCAPLUS ABB=ON PLU=ON L7 AND ASSAY?
L9 3 SEA FILE=HCAPLUS ABB=ON PLU=ON L8 AND (CHIP OR ?ARRAY? OR
SURFACE OR ?SILAN? OR GLASS)
L10 24 SEA FILE=HCAPLUS ABB=ON PLU=ON L8 NOT L9
L11 1 SEA FILE=HCAPLUS ABB=ON PLU=ON L10 AND LIGAND(2A) BIND?
L12 4 SEA FILE=HCAPLUS ABB=ON PLU=ON L9 OR L11
L13 4 SEA FILE=REGISTRY ABB=ON PLU=ON (127361-24-4/B1 OR 217962-21-
5/B1 OR 250154-17-7/B1 OR 80451-05-4/B1)
L14 2 SEA FILE=HCAPLUS ABB=ON PLU=ON L12 AND L13
L15 4 SEA FILE=HCAPLUS ABB=ON PLU=ON L12 OR L14
L16 509 SEA FILE=HCAPLUS ABB=ON PLU=ON (?PROTEIN? OR ?PEPTID?) (2A) CHI
P
L17 1 SEA FILE=HCAPLUS ABB=ON PLU=ON L16 AND L6
L18 24061 SEA FILE=HCAPLUS ABB=ON PLU=ON (?PROTEIN? OR ?PEPTID?) (5A) (?I
MMOB? OR ATTACH? OR SPAN? OR FIX OR FIXED OR FIXING)
L19 18 SEA FILE=HCAPLUS ABB=ON PLU=ON L6 AND L18
L20 9 SEA FILE=HCAPLUS ABB=ON PLU=ON L19 AND (?ARRAY? OR SURFACE
OR ?SILAN? OR GLASS OR ?MEMBRAN?)
L21 9 SEA FILE=HCAPLUS ABB=ON PLU=ON L20 NOT L15
L22 10 SEA FILE=HCAPLUS ABB=ON PLU=ON L17 OR L21

10 cites

=> d ibib abs 1

L22 ANSWER 1 OF 10 HCPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 2001:126377 HCPLUS
 DOCUMENT NUMBER: 134:293189
 TITLE: Biosynthesis of surfactant protein C: characterization
 of aggresome formation by EGFP chimeras containing
 propeptide mutants lacking conserved cysteine residues
 AUTHOR(S): Kabore, Albert F.; Wang, Wen-Jing; Russo,
 Scott J.; Beers, Michael F.
 CORPORATE SOURCE: Lung Epithelial Cell Biology Laboratories, Pulmonary
 and Critical Care Division, Department of Medicine,
 University of Pennsylvania School of Medicine,
 Philadelphia, PA, 19104, USA
 SOURCE: Journal of Cell Science (2001), 114(2), 293-302
 CODEN: JNCSAI; ISSN: 0021-9533
 PUBLISHER: Company of Biologists Ltd.
 DOCUMENT TYPE: Journal
 LANGUAGE: English

AB Surfactant protein C (SP-C) is a lung-specific secreted protein, which is synthesized as a 21-kDa propeptide (SP-C21) and then proteolytically processed as a bitopic **transmembrane** protein in subcellular compartments distal to the medial Golgi to produce a 3.7 kDa mature form. We have shown that initial processing of SP-C21 involves two endoproteolytic cleavages of the C terminus and that truncation of nine amino acids from the C-flanking peptide resulted in retention of mutant protein in proximal compartments. Because these truncations involved removal of a conserved cysteine residue (Cys186), we hypothesized that intraluminal disulfide-mediated folding of the C terminus of SP-C21 is required for intracellular trafficking. To test this, cDNA constructs encoding heterologous fusion proteins consisting of enhanced green fluorescent **protein** (EGFP) attached to the N terminus of wild-type rat proSP-C (EGFP/SP-C1-194), C-terminally deleted proSP-C (EGFP/SP-C1-185; EGFP/SP-C1-191) or point mutations of conserved cysteine residues (EGFP/SP-CC122G; EGFP/SP-CC186G; or EGFP/SP-CC122/186G) were transfected into A549 cells. Fluorescence microscopy revealed that transfected EGFP/SP-C1-194 and EGFP/SP-C1-191 were expressed in a punctate pattern within CD-63 pos., EEA-1 neg. cytoplasmic vesicles. In contrast, EGFP/SP-C1-185, EGFP/SP-CC122G, EGFP/SP-CC186G and EGFP/SP-CC122/186G were expressed but retained in a juxtanuclear compartment that stained for ubiquitin and that contained .gamma.-tubulin and vimentin, consistent with expression in aggresomes. Treatment of cells transfected with mutant proSP-C with the proteasome inhibitor lactacysteine enhanced aggresome formation, which could be blocked by coincubation with nocodazole. Western blots using a GFP antibody detected a single form in lysates of cells transfected with EGFP/SP-C cysteine mutants, without evidence of smaller degrdn. fragments. We conclude that residues Cys122 and Cys186 of proSP-C are required for proper post-translational trafficking. Mutation or deletion of one or both of these residues results in misfolding with mistargeting of unprocessed mutant protein, leading to formation of stable aggregates within aggresomes.

REFERENCE COUNT: 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d ibib abs 2

L22 ANSWER 2 OF 10 HCPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 2000:621123 HCPLUS
 DOCUMENT NUMBER: 133:314134
 TITLE: Method for Fabricating Supported Bilayer Lipid
Membranes on Gold
 AUTHOR(S): Lahiri, Joydeep; Kalal, Peter;
 Frutos, Anthony G.; Jonas, Steven J.;
 Schaeffler, Robert
 CORPORATE SOURCE: Biochemistry and Surfaces and Interfaces Core
 Technologies Science and Technology Division, Corning
 Incorporated, Corning, NY, 14831, USA
 SOURCE: Langmuir (2000), 16(20), 7805-7810
 CODEN: LANGD5; ISSN: 0743-7463
 PUBLISHER: American Chemical Society
 DOCUMENT TYPE: Journal
 LANGUAGE: English

AB The paper describes the synthesis of **surfaces** based on self-assembled monolayers (SAMs) of alkanethiolates on Au that were designed to support the adsorption of bilayer lipid **membranes**, and the feasibility of using these **surfaces** for making high-d. **arrays** (HDAs) of **membranes**. The synthesis involved (i) the formation of SAMs of 16-mercaptophexadecanoic acid (MHA) on Au; (ii) the activation of the carboxylic acid groups to interchain anhydrides; (iii) the treatment of the reactive **surfaces** with Brij-76 ($C_{18}H_{37}(OCH_2CH_2)_{n-10}OH$) (1) or Brij-76-amine (2) ($C_{18}H_{37}(OCH_2CH_2)_{n-10}NH_2$); and (iv) the formation of supported **membranes** by incubation or printing of the lipids. The oligo(ethylene glycol) moiety of the anchor lipid provides a hydrophilic spacer between the **surface** and the adsorbed lipid to enable the incorporation of **membrane-spanning proteins** with extra-**membrane** domains. Data from IR spectroscopy confirmed the coupling of 2 to the **surface** through the formation of peptide bonds. Ellipsometric measurements showed an increase of .apprx.15 .ANG. in the thickness of the SAM after coupling to 2; this observation suggests that .apprx.25% of the carboxylic acid groups of the MHA-SAM are derivatized with Brij groups. The yields for coupling of 1 were .apprx.40% of that obsd. for 2. Expts. using **surface** plasmon resonance (SPR) were consistent with the binding of lipid bilayers to the Brij-derivatized **surface**, although alternative structures for the supported lipids cannot be ruled out; by contrast, SAMs of hexadecanethiolate on Au bind lipid monolayers. Biospecific binding of neutravidin was obsd. on supported **membranes** incorporating biotinylated lipids. HDAs of lipids were made by printing lipids onto the Brij-derivatized **surfaces** using a quill-pin printer. Fluorescence microscopy indicated that the printed lipids remained confined to the printed areas; these observations demonstrate the applicability of using com. HDA printers for generating high-d. **membrane arrays** on the Brij-modified **surfaces**.

REFERENCE COUNT: 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d ibib abs 3

L22 ANSWER 3 OF 10 HCPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:617374 HCPLUS
TITLE: Novel chemistry for parallel synthesis of microarrays
of oligonucleotides and peptides using photogenerated
acids.
AUTHOR(S): Gao, Xiaolian; LeProust, Eric M.; Pellois, Jean
Philippe; Yu, Peilin; Zhang, Hua; Wang, Wei;
Zhou, Xiaochuan
CORPORATE SOURCE: Department of Chemistry, University of Houston,
Houston, TX, 77204-5641, USA
SOURCE: Book of Abstracts, 218th ACS National Meeting, New
Orleans, Aug. 22-26 (1999), MEDI-003. American
Chemical Society: Washington, D. C.
CODEN: 67ZJA5
DOCUMENT TYPE: Conference; Meeting Abstract
LANGUAGE: English
AB We present novel soln. chem. that is suitable for parallel synthesis of
biomols. and a broad range of org. mols. completed with simple and
efficient operations. The novelty of the chem. described herein lies in
its use of photo-generated reagents to affect otherwise conventional chem.
The synthesis of oligonucleotides using photo-generated acids has been
established (Gao et al., J. 120, 12688-12689). In this presentation, the
synthesis of peptides using photo-generated acids and combinatorial
screening using the addressable chip technol. will be described. Our method
using photo-generated reagents rather than conventional reagents provides
an optical switch to allow light-initiation of conventional reactions.
This new soln. chem. takes the advantage of well-established synthetic
procedures and materials and therefore, promises simple, flexible, high
quality and low cost parallel synthesis of microarrays of
oligonucleotides, peptides and diverse mol. sequences. Parallel synthesis
of microarrays of biomols. on planar solid surfaces is a highly efficient
means for generating high-d. biochips such as DNA/RNA- and peptide
-chips, which are powerful high throughput anal. tools.
However, the current technologies have significant drawbacks and are not
applicable to a vast majority of research and applied labs. in related
fields. Our novel chem. forms foundation for a programmable maskless
automated microarray synthesizer that is cost-affordable and tech.
manageable by most labs. interested in biochips. The availability of such
an instrument would significantly accelerate the processes of gene anal.
and development of gene-based applications.

=> d ibib abs 4

L22 ANSWER 4 OF 10 HCAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1999:17704 HCAPLUS
 DOCUMENT NUMBER: 130:234184
 TITLE: A Strategy for the Generation of **Surfaces**
 Presenting Ligands for Studies of Binding Based on an
 Active Ester as a Common Reactive Intermediate: A
Surface Plasmon Resonance Study
 AUTHOR(S): Lahiri, Joydeep; Isaacs, Lyle; Tien, Joe;
 Whitesides, George M.
 CORPORATE SOURCE: Department of Chemistry and Chemical Biology, Harvard
 University, Cambridge, MA, 02138, USA
 SOURCE: Anal. Chem. (1999), 71(4), 777-790
 CODEN: ANCHAM; ISSN: 0003-2700
 PUBLISHER: American Chemical Society
 DOCUMENT TYPE: Journal
 LANGUAGE: English

AB This paper describes the **immobilization** of ten **proteins** and two low-mol.-wt. ligands on mixed selfassembled monolayers (SAMs) of alkanethiolates on gold generated from the tri(ethylene glycol)-terminated thiol 1 ($\text{HS}(\text{CH}_2)_{11}(\text{OCH}_2\text{CH}_2)_3\text{OH}$) (χ_1 .(1) = 1.0-0.0) and the longer, carboxylic acid-terminated thiol 2 ($\text{HS}(\text{CH}_2)_{11}(\text{OCH}_2\text{CH}_2)_6\text{OCH}_2\text{CO}_2\text{H}$) (χ_1 .(2) = 0.0-1.0). The immobilization was achieved by a two-step procedure: generation of reactive N-hydroxysuccinimidyl esters from the carboxylic acid groups of 2 in the SAM and coupling of these groups with amines on the protein or ligand. Because this method involves a common reactive intermediate that is easily prep'd., it provides a convenient method for attaching ligands to SAMs for studies using **surface** plasmon resonance spectroscopy (and, in principle, other bioanal. methods that use derivatized SAMs on gold, silver, and other **surfaces**). These SAMs were resistant to nonspecific adsorption of proteins having a wide range of mol. wts. and isoelec. points. The pH of the coupling buffer, the concn. of protein, the ionic strength of the soln. of protein, and the mol. wt. of the protein all influenced the amt. of the **protein** that was **immobilized**. For the proteins investigated in detail-carbonic anhydrase and lysozyme-the highest quantities of **immobilized proteins** were obtained when using a low ionic strength soln. at a value of pH approx. one unit below the isoelec. point (pI) of the protein, at a concn. of .apprx.0.5 mg mL⁻¹. Comparisons of the kinetic and thermodn. consts. describing binding of carbonic anhydrase and vancomycin to immobilized benzenesulfonamide and N-.alpha.-Ac-Lys-D-Ala-D-Ala groups, resp., on mixed SAMs (by methods described in this paper) and in the carboxymethyl dextran matrix of com. available substrates yielded (for these systems) essentially indistinguishable values of Kd, koff, and kon.

REFERENCE COUNT: 65 THERE ARE 65 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d ibib abs 5

L22 ANSWER 5 OF 10 HCPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:7458 HCPLUS
DOCUMENT NUMBER: 130:179567
TITLE: Site-specific immobilization of Fab' fragments of goat
antihuman IgG on quartz **surfaces**
AUTHOR(S): Qian, Weiping; Fang, Yu; Song, Zhendong;
Liang, Bingjie; Yu, Wei
CORPORATE SOURCE: National Laboratory of Molecular and Biomolecular
Electronics, Southeast University, Nanjing, 210096,
Peop. Rep. China
SOURCE: Supramol. Sci. (1998), 5(5-6), 701-703
CODEN: SUSCFX; ISSN: 0968-5677
PUBLISHER: Elsevier Science Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB This paper presents a strategy for immobilizing biomols. onto a solid
surface. We used the free thiol group directing method to
immobilize Fab' fragments to the MPTMS derived quartz substrate, and
results of X-ray photoelectron spectroscopic studies on the binding of
MTPMS and Fab' fragments on the quartz **surfaces** are reported.
We also prep'd. a new type of immuno-labeled protein which was the rare
earth element labeled antigen (human IgG) to recognize Fab' fragment.
Four characteristic peaks has been detd. on the **surface**
specifically bound human IgG labeled Tb³⁺.
REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d ibib abs 6

L22 ANSWER 6 OF 10 HCPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:173428 HCPLUS
DOCUMENT NUMBER: 126:220777
TITLE: Recognition of [³H]oxytocin-receptor complex using
immobilized antisense peptide
AUTHOR(S): Lu, Fengxian; Wang, Wei; Tang, Te
CORPORATE SOURCE: Lab. Physiol. Pathol., Tianjin Med. Univ., Tianjin,
300070, Peop. Rep. China
SOURCE: Zhongguo Bingli Shengli Zazhi (1996), 12(3), 239-243
CODEN: ZBSZEB; ISSN: 1000-4718
PUBLISHER: Jinan Daxue
DOCUMENT TYPE: Journal
LANGUAGE: Chinese
AB An antisense peptide for oxytocin, whose sequence was encoded in an
antisense strand of DNA corresponding to the 12 N-terminal residues of
bovine oxytocin (OT) and its binding protein, neurophysin I, a region that
included the OT sequence at residues 1-9 was prep'd. The peptide
selectively bound to OT and the solubilized complex of OT with receptor
protein in plasma **membranes** from rat mammary gland after
parturition. The mol. wt. of the OT receptor protein was 65 kDa. The
results suggest this peptide might be used as a tool for affinity
chromatog.

=> d ibib abs 7

L22 ANSWER 7 OF 10 HCPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1992:191866 HCPLUS
DOCUMENT NUMBER: 116:191866
TITLE: Differential glycosylation and cell **surface**
expression of lysosomal **membrane**
glycoproteins in sublines of a human colon cancer
exhibiting distinct metastatic potentials
AUTHOR(S): Saitoh, Osamu; Wang, Wei Chun; Lotan,
Reuben; Fukuda, Minoru
CORPORATE SOURCE: Cancer Res. Cent., La Jolla Cancer Res. Found., La
Jolla, CA, 92037, USA
SOURCE: J. Biol. Chem. (1992), 267(8), 5700-11
CODEN: JBCHA3; ISSN: 0021-9258
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Changes in the glycosylation of asparagine-linked oligosaccharides have
been shown in various tumor cells, including human colon cancer. Attempts
were made to elucidate the difference in Asn-linked oligosaccharides
attached to lysosomal membrane glycoproteins
isolated from sublines of human colon carcinoma exhibiting high and low
metastatic potentials in nude mice. Lysosomal **membrane**
glycoproteins (lamp) 1 and 2 were immunopptd. from the cells after
labeling with radioactive sugars, and the glycopeptides prep'd. were
fractionated by serial lectin affinity chromatog. employing immobilized
Con A, Datura stramonium agglutinin, and tomato lectin. Comparison of
Asn-linked oligosaccharides from the different colonic carcinoma cells
revealed the following features. First, the highly metastatic carcinoma
cells express more poly-N-acetyllactosamyl side chains with branched
galactose residues than cells with low metastatic potential. Second,
sialylation is more significant in the highly metastatic carcinoma cells
than in the poorly metastatic ones. Conversely, N-acetyllactosamine units
are less fucosylated in the highly metastatic cells than in poorly
metastatic cells. These structural changes were apparently caused by the
increase in sialyltransferase and the decrease in .alpha.1.fwdarw.3
fucosyltransferase in the highly metastatic cells. The results also
suggest that highly metastatic carcinoma cells express more sialyl Lex
structures at the termini of poly-N-acetyllactosamyl side chains than
poorly metastatic carcinoma cells. Further, highly metastatic cells were
found to express more lamp-1 and lamp-2 on the cell **surface**.
These results were found to be correlated to the increased expression of
sialyl Lex structures with high affinity binding of anti-sialyl Lex
antibody on highly metastatic cells. Increased expression of sialyl Lex
in the poly-N-acetyllactosamines of the cell **surface** may
contribute to the metastatic behavior of the cells, assuming that this
structure can serve as a better ligand for selectins present on
endothelial cells and platelets.

=> d ibib abs 8

L22 ANSWER 8 OF 10 HCPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1992:124233 HCPLUS
DOCUMENT NUMBER: 116:124233
TITLE: Sensor of glucose oxidase immobilized in Bombyx mori
silk fibroin **membrane**
AUTHOR(S): Shao, Zhengzhong; Fang, Yue; Yu, Tongyin;
Deng, Jiaqi
CORPORATE SOURCE: Dep. Mater. Sci., Fudan Univ., Shanghai, 200433, Peop.
Rep. China
SOURCE: Gaodeng Xuexiao Huaxue Xuebao (1991), 12(6), 847-8
CODEN: KTHPDM; ISSN: 0251-0790
DOCUMENT TYPE: Journal
LANGUAGE: Chinese
AB Glucose oxidase (I) can be immobilized in B. mori silk fibroin
membrane due to the structural transition of the silk fibroin
membrane from random coil to antiparallel .beta.-sheet induced by
MeOH treatment. The glucose-selective sensor was made of this I
immobilized fibroin **membrane**. The calibration curve, response
time, recovery, and thermal and storage stability were detd.

=> d ibib abs 9

L22 ANSWER 9 OF 10 HCPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1991:676542 HCPLUS
DOCUMENT NUMBER: 115:276542
TITLE: The poly-N-acetyllactosamines attached to
lysosomal **membrane glycoproteins**
are increased by the prolonged association with the
Golgi complex
AUTHOR(S): Wang, Wei Chun; Lee, Ni; Aoki, Daisuke;
Fukuda, Michiko N.; Fukuda, Minoru
CORPORATE SOURCE: La Jolla Cancer Res. Found., Cancer Res. Cent., La
Jolla, CA, 92037, USA
SOURCE: J. Biol. Chem. (1991), 266(34), 23185-90
CODEN: JBCHA3; ISSN: 0021-9258
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The poly-N-acetyllactosamines on neutrophils and monocytes have been shown
to serve as ligands for various selectins present on endothelial cells and
platelets. To understand the reason why only certain glycoproteins can be
modified by poly-N-acetyllactosamine, the authors utilized 21.degree.
incubation conditions, which had previously been shown to cause the
accumulation of glycoproteins at the trans-Golgi. HL-60 cells were
labeled with [3H]galactose at 21 or 37.degree. for 6 or 24 h, and the
lysosomal **membrane glycoproteins** lamp-1 and lamp-2 were
immunopptd. On examn. by SDS-PAGE, each lamp from HL-60 cells incubated
at 21.degree. exhibited a much broader, slower migrating band than that
isolated from the cells incubated at 37.degree.. The no. of N-glycans
contg. poly-N-acetyllactosamine, estd. by their binding to tomato lectin
column, increased .apprx.30-50% after incubation at 21.degree., compred to
incubation at 37.degree.. The anal. of oligosaccharides released by
endo-.beta.-galactosidase digestion demonstrates that the amt. of side
chains contg. .gtoreq.3 N-acetyllactosamine repeats increased .apprx.100%
after incubation at 21.degree., and methylation anal. confirmed these
results. The same anal. and the results obtained by ion-exchange
chromatog. also provided evidence that the N-glycans of lamps are
sialylated at 21.degree. as much as at 37.degree.. Pulse-chase expts.
using [35S]methionine labeling indicated that the time necessary for
processing of lamps is much longer at 21.degree. than at 37.degree..
These results therefore indicate that incubation at 21.degree. cause the
lamps to reside longer within the Golgi complex, and such longer residence
allows lamps to acquire more polylactosaminoglycan. These results also
suggest that the time necessary for moving through the Golgi complex is a
crit. factor for poly-N-acetyllactosamine formation.

=> d ibib abs 10

L22 ANSWER 10 OF 10 HCPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1990:589612 HCPLUS
DOCUMENT NUMBER: 113:189612
TITLE: Granulocytic differentiation of HL-60 cells is associated with increase of poly-N-acetyllactosamine in Asn-linked oligosaccharides **attached** to human lysosomal **membrane glycoproteins**
AUTHOR(S): Lee, Ni; Wang, Wei Chun; Fukuda, Minoru
CORPORATE SOURCE: Cancer Res. Cent., La Jolla Cancer Res. Found., La Jolla, CA, 92037, USA
SOURCE: J. Biol. Chem. (1990), 265(33), 20476-87
CODEN: JBCHA3; ISSN: 0021-9258
DOCUMENT TYPE: Journal
LANGUAGE: English
AB HL-60 cells were induced to differentiate into granulocytic cells by DMSO, and structures of Asn-linked oligosaccharides **attached** to lysosomal **membrane glycoproteins** (lamp-1 and lamp-2) were elucidated before and after differentiation. Lamp-1 and lamp-2 were immunopptd. from the cells after labeling with radioactive sugars, and glycopeptides were prep'd. The structures of glycopeptides obtained after serial lectin-affinity chromatog. were elucidated by endo-.beta.-galactoside and methylation anal. Glycopeptides bound to tomato lectin-Sepharose were tetraantennary oligosaccharides that contain two or three poly-N-acetyllactosaminyl chains, of which one side chain contains three or more N-acetyllactosaminyl repeats, whereas those bound to Datura stramonium agglutinin-Sepharose were tetraantennary oligosaccharides contg. one or two short poly-N-acetyllactosaminyl side chains. Glycopeptides that were not bound to Con A, tomato lectin, or D. stramonium agglutinin were triantennary oligosaccharides with a negligible amt. of poly-N-acetyllactosaminyl side chains. Comparison of Asn-linked oligosaccharides from undifferentiated and differentiated HL-60 cells reveals the following features. First, the no. of Asn-linked oligosaccharides contg. poly-N-acetyllactosaminyl side chains increases dramatically with a concomitant decrease in less complex Asn-linked oligosaccharides after differentiation. Second, the no. of poly-N-acetyllactosaminyl side chains per Asn-linked oligosaccharides increases. These increases in poly-N-acetyllactosamine were assocd. with increased activity of UDP-GlcNAc:.beta.-D-Gal-.beta.1 .fwdarw. 3-N-acetylglucosaminyltransferase extension enzyme, a key enzyme in the formation of poly-N-acetyllactosamines. The increased amt. of poly-N-acetyllactosamine in lamp-1 and lamp-2 resulted in longer half-lives of lamp-1 and lamp-2 in differentiated HL-60 cells. These results suggest strongly that the differentiation of HL-60 cells into more phagocytic cells is assocd. with an increase in the complexity of Asn-linked oligosaccharides **attached** to lysosomal **membrane glycoproteins**, which in turn may play a role in stabilizing lysosomes.